Your SELECT statement is:
s epha2
Items File
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41 5: Biosis Previews(R)_1969-2002/Jul W3
31 34: SciSearch(R) Cited Ref Sci_1990-2002/Jul W4
3 35: Dissertation Abs Online_1861-2002/Jun
24 71: ELSEVIER BIOBASE_1994-2002/Jul W3
27 73: EMBASE_1974-2002/Jul W3
2 77: Conference Papers Index_1973-2002/Jul
1 94: JICST-EPlus_1985-2002/Jun W1
4 98: General Sci Abs/Full-Text_1984-2002/Jun
1 135: NewsRx Weekly Reports_1995-2002/Jul W3
11 144: Pascal_1973-2002/Jul W4
1 149: TGG Health&Wellness DB(SM)_1976-2002/Jul W2
47 155: MEDLINE(R)_1966-2002/Jul W3
3 156: ToxFile_1965-2002/Jul W2
26 159: Cancerlit_1975-2002/Jun
1 172: EMBASE Alert_2002/Jul W4
3 266: FEDRIP_2002/Jun
25 399: CA SEARCH(R)_1967-2002/UD=13705

Set	Items	Description
S1	0	SPHA2
S2	114	EPHA2
S3	54	S2 NOT PY=>2000
S4	33	RD (unique items)
S5	13	S4 AND (CANCER? OR TUMOR?)

5/9/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12329023 BIOSIS NO.: 200000082525
Overexpression of EphA2 **in metastatic** cancer **cells: A role for Ras signaling.**
AUTHOR: Walker-Daniels Jennifer L(a); Zantek Nicole D(a); Azimi Minou(a); Kinch Michael S(a)
AUTHOR ADDRESS: (a)Purdue University, 1246 Lynn Hall, West Lafayette, IN, 47907-1246**USA
JOURNAL: Molecular Biology of the Cell 10 (SUPPL.):p427a Nov., 1999
CONFERENCE/MEETING: 39th Annual Meeting of the American Society for Cell Biology Washington, D.C., USA December 11-15, 1999
SPONSOR: The American Society for Cell Biology
ISSN: 1059-1524
RECORD TYPE: Citation
LANGUAGE: English
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; **Tumor** Biology
ORGANISMS: PARTS ETC: metastatic **cancer** cells
DISEASES: breast **cancer** --neoplastic disease, reproductive system disease/female; epithelial **cancers** --neoplastic disease; prostate **cancer** --neoplastic disease, reproductive system disease/male, urologic disease
CHEMICALS & BIOCHEMICALS: **EphA2** --overexpression; Ras
MISCELLANEOUS TERMS: Meeting Abstract
ALTERNATE INDEXING: Breast Neoplasms (MeSH); Prostatic Neoplasms (MeSH)
CONCEPT CODES:
24002 Neoplasms and Neoplastic Agents-General
02502 Cytology and Cytochemistry-General
10060 Biochemical Studies-General
13002 Metabolism-General Metabolism; Metabolic Pathways
16501 Reproductive System-General; Methods
15501 Urinary System and External Secretions-General; Methods
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals

5/9/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12301863 BIOSIS NO.: 200000059730

Overexpression of the EphA2 **tyrosine kinase in prostate** cancer .
AUTHOR: Walker-Daniels J; Coffman K; Azimi M; Rhim J S; Bostwick D G; Snyder P; Kerns B J; Waters D J; Kinch M S(a)
AUTHOR ADDRESS: (a)Department of Basic Medical Sciences, 1246 Lynn Hall, West Lafayette, IN**USA
JOURNAL: Prostate 41 (4):p275-280 Dec. 1, 1999
ISSN: 0270-4137
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: BACKGROUND: Molecules that are highly expressed by human prostate **cancers** may serve as therapeutically relevant targets or **tumor** markers. Tyrosine kinases are frequently overexpressed in metastatic **tumor** cells and this prompted us to screen for tyrosine kinases that are overexpressed in prostate **cancer** cells. METHODS: Expression levels of the **EphA2** receptor tyrosine kinase were determined by Western blot analysis in canine and human prostate **cancer** cell lines and in immortalized and transformed variants of 267B1 prostatic epithelial cells. **EphA2** levels in benign human prostate and prostate **cancers** were also determined in formalin-fixed, paraffin-embedded tissues using immunohistochemical staining. RESULTS: Metastatic prostate **cancer** cells overexpressed **EphA2** by 10-100 fold as compared with non-invasive prostatic epithelial cells. **EphA2** immunoreactivity in vivo was also significantly greater in human prostate **cancers** as compared with benign prostate epithelium. CONCLUSIONS: The **EphA2** receptor tyrosine kinase is differentially expressed in human and canine prostate **cancer** cell lines and overexpressed in human prostate **cancers** as compared with benign prostate tissues. Metastasis-derived canine prostate carcinoma cell lines overexpress **EphA2** and may provide pre-clinical models to further evaluate the role of **EphA2** in prostate carcinogenesis. Further investigations are needed to determine the utility of **EphA2** as a **tumor** marker and a novel target in human prostate **cancer** .

DESCRIPTORS:
MAJOR CONCEPTS: Enzymology (Biochemistry and Molecular Biophysics); Reproductive System (Reproduction); **Tumor** Biology
BIOSYSTEMATIC NAMES: Canidae--Carnivora, Mammalia, Vertebrata, Chordata, Animalia; Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: 267B1 cell line (Hominidae)--human prostate epithelial **tumor** cells; BC-1 cell line (Canidae)--canine prostate carcinoma cells; BF-2 cell line (Canidae)--canine prostate carcinoma cells; CF-3 cell line (Canidae)--canine prostate carcinoma cells; GN-4 cell line (Canidae)--canine prostate carcinoma cells; TR5P cell line (Canidae)--canine prostate carcinoma cells; TR6LM cell line (Canidae)--canine prostate carcinoma cells
ORGANISMS: PARTS ETC: prostate epithelium--reproductive system
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Carnivores; Chordates ; Humans; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Primates; Vertebrates
CHEMICALS & BIOCHEMICALS: **EphA2** receptor tyrosine kinase--expression, **tumor** marker
METHODS & EQUIPMENT: Western blot--analytical method, detection/labeling techniques, gene mapping; immunohistochemistry--analytical method
CONCEPT CODES:
24002 Neoplasms and Neoplastic Agents-General
10802 Enzymes-General and Comparative Studies; Coenzymes
15501 Urinary System and External Secretions-General; Methods
16501 Reproductive System-General; Methods
BIOSYSTEMATIC CODES:
85765 Canidae
86215 Hominidae

5/9/3 (Item 3 from file: 5)

12190446 BIOSIS NO.: 199900485295
E-cadherin regulates the function of the EphA2 **receptor tyrosine kinase.**
AUTHOR: Dodge Zantek Nicole; Azimi Minoudokht; Fedor-Chaiken Mary; Wang Bingcheng; Brackenbury Robert; Kinch Michael S(a)
AUTHOR ADDRESS: (a)Department of Basic Medical Sciences, Purdue University, West Lafayette, IN, 47907-1246**USA
JOURNAL: Cell Growth & Differentiation 10 (9):p629-638 Sept., 1999
ISSN: 1044-9523
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: **EphA2** is a member of the Eph family of receptor tyrosine kinases, which are increasingly understood to play critical roles in disease and development. We report here the regulation of **EphA2** by E-cadherin. In nonneoplastic epithelia, **EphA2** was tyrosine-phosphorylated and localized to sites of cell-cell contact. These properties required the proper expression and functioning of E-cadherin. In breast **cancer** cells that lack E-cadherin, the phosphotyrosine content of **EphA2** was decreased, and **EphA2** was redistributed into membrane ruffles. Expression of E-cadherin in metastatic cells restored a more normal pattern of **EphA2** phosphorylation and localization. Activation of **EphA2** , either by E-cadherin expression or antibody-mediated aggregation, decreased cell-extracellular matrix adhesion and cell growth. Altogether, this demonstrates that **EphA2** function is dependent on E-cadherin and suggests that loss of E-cadherin function may alter neoplastic cell growth and adhesion via effects on **EphA2** .

DESCRIPTORS:
MAJOR CONCEPTS: Development; Enzymology (Biochemistry and Molecular Biophysics); Membranes (Cell Biology); **Tumor** Biology
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: human (Hominidae)
ORGANISMS: PARTS ETC: breast cells--reproductive system; cell membrane; extracellular matrix; mammary epithelial cells--endocrine system
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Humans; Mammals; Primates; Vertebrates
DISEASES: carcinoma--neoplastic disease
CHEMICALS & BIOCHEMICALS: E-cadherin; **EphA2** --phosphorylation, receptor tyrosine kinase, regulation
METHODS & EQUIPMENT: western blotting--analytical method
MISCELLANEOUS TERMS: cell growth
ALTERNATE INDEXING: Carcinoma (MeSH)
CONCEPT CODES:
24002 Neoplasms and Neoplastic Agents-General
02508 Cytology and Cytochemistry-Human
03508 Genetics and Cytogenetics-Human
10060 Biochemical Studies-General
10802 Enzymes-General and Comparative Studies; Coenzymes
BIOSYSTEMATIC CODES:
86215 Hominidae

5/9/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11941339 BIOSIS NO.: 199900187448
Regulation of the EphA2 **receptor tyrosine kinase by estrogen and myc.**
AUTHOR: Zantek N D; Zelinski D; Peters M A; Taparowsky E J; Kinch M S
AUTHOR ADDRESS: Purdue Univ., West Lafayette, IN 47907**USA
JOURNAL: Proceedings of the American Association for Cancer Research Annual Meeting 40p687 March, 1999
CONFERENCE/MEETING: 90th Annual Meeting of the American Association for Cancer Research Philadelphia, Pennsylvania, USA April 10-14, 1999
SPONSOR: American Association for Cancer Research

ISSN: 0197-016X
RECORD TYPE: Citation
LANGUAGE: English
REGISTRY NUMBERS: 80449-02-1: TYROSINE KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Enzymology (Biochemistry and Molecular Biophysics); **Tumor** Biology
DISEASES: breast **cancer** --neoplastic disease, reproductive system disease/female
CHEMICALS & BIOCHEMICALS: c-myc protein--transcription factor; estrogen; **EphA2** receptor tyrosine kinase--regulation
MISCELLANEOUS TERMS: Meeting Abstract
ALTERNATE INDEXING: Breast Neoplasms (MeSH)
CONCEPT CODES:
10802 Enzymes-General and Comparative Studies; Coenzymes
10060 Biochemical Studies-General
16501 Reproductive System-General; Methods
24002 Neoplasms and Neoplastic Agents-General
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals

5/9/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11780721 BIOSIS NO.: 199900026830
Epithelial cell kinase (ECK/ EPHA2 **) regulation in breast** cancer .
AUTHOR: Zantek Nicole Dodge(a); Fedor-Chaiken Mary; Brackenbury Robert; Kinch Michael S
AUTHOR ADDRESS: (a)Dep. Basic Med. Sci., Purdue Univ., West Lafayette, IN 47907**USA
JOURNAL: Molecular Biology of the Cell 9 (SUPPL.):p134A Nov., 1998
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
RECORD TYPE: Citation
LANGUAGE: English
REGISTRY NUMBERS: 9031-44-1: KINASE; 80449-02-1: TYROSINE KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Enzymology (Biochemistry and Molecular Biophysics); **Tumor** Biology
DISEASES: breast **cancer** --neoplastic disease, reproductive system disease/female
CHEMICALS & BIOCHEMICALS: E-cadherin; Epithelial Cell Kinase {ECK/ **EphA2** }--Eph family tyrosine kinase, breast **cancer** progression marker
MISCELLANEOUS TERMS: Meeting Abstract
ALTERNATE INDEXING: Breast Neoplasms (MeSH)
CONCEPT CODES:
24002 Neoplasms and Neoplastic Agents-General
02502 Cytology and Cytochemistry-General
10060 Biochemical Studies-General
10802 Enzymes-General and Comparative Studies; Coenzymes
16501 Reproductive System-General; Methods
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
?5/9/6 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10446836 99439331 PMID: 10511313
E-cadherin regulates the function of the EphA2 **receptor tyrosine kinase.**
Zantek N D; Azimi M; Fedor-Chaiken M; Wang B; Brackenbury R; Kinch M S
Department of Basic Medical Sciences and Purdue Cancer Center, Purdue University, West Lafayette, Indiana 47907, USA.
Cell growth & differentiation : the molecular biology journal of the American Association for Cancer Research (UNITED STATES) Sep 1999, 10 (9) p629-38, ISSN 1044-9523 Journal Code: 9100024

Contract/Grant No.: AR44713; AR; NIAMS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

EphA2 is a member of the Eph family of receptor tyrosine kinases, which are increasingly understood to play critical roles in disease and development. We report here the regulation of **EphA2** by E-cadherin. In nonneoplastic epithelia, **EphA2** was tyrosine-phosphorylated and localized to sites of cell-cell contact. These properties required the proper expression and functioning of E-cadherin. In breast **cancer** cells that lack E-cadherin, the phosphotyrosine content of **EphA2** was decreased, and **EphA2** was redistributed into membrane ruffles. Expression of E-cadherin in metastatic cells restored a more normal pattern of **EphA2** phosphorylation and localization. Activation of **EphA2** , either by E-cadherin expression or antibody-mediated aggregation, decreased cell-extracellular matrix adhesion and cell growth. Altogether, this demonstrates that **EphA2** function is dependent on E-cadherin and suggests that loss of E-cadherin function may alter neoplastic cell growth and adhesion via effects on **EphA2** .

5/9/7 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10431502 99434114 PMID: 10502726
Up-regulation of ephrin-A1 during melanoma progression.
Easty D J; Hill S P; Hsu M Y; Fallowfield M E; Florenes V A; Herlyn M; Bennett D C
St. George's Hospital Medical School, London, UK. david.easty@ucd.ie
International journal of cancer. Journal international du cancer (UNITED STATES) Oct 22 1999, 84 (5) p494-501, ISSN 0020-7136 Journal Code: 0042124
Contract/Grant No.: CA 25874; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Ephrin-A1, formerly called B61, is a new melanoma growth factor; it is angiogenic and chemoattractant for endothelial cells. EPH-A2, or ECK (a receptor for ephrin-A1), is ectopically expressed in most melanoma cell lines; the pathology where this expression is first manifested and the possible role of the receptor in **tumor** progression are unknown. To determine these, we studied the expression of this ligand and receptor in biopsies of benign and malignant melanocytic lesions. EPH-A2 was not detected in normal melanocytes, benign compound nevi or advanced melanomas, though it was found in 2 of 9 biopsies of malignant melanoma in situ. Ephrin-A1 was present in occasional early lesions and in advanced primary melanomas (43%) and metastatic melanomas (67%). Expression of ephrin-A1 was induced in melanoma cells by pro-inflammatory cytokines. Our findings are consistent with 2 possible roles for ephrin-A1 in melanoma development: it may promote melanocytic cell growth or survival and induce vascularization in advanced melanomas. Both effects may be potentiated by inflammatory responses. Our data are consistent with earlier observations that an inflammatory infiltrate is associated with poor prognosis in thin primary melanomas. Copyright 1999 Wiley-Liss, Inc.

5/9/8 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09635105 98057825 PMID: 9396043
Overexpression of protein tyrosine kinases in human esophageal cancer .
Nemoto T; Ohashi K; Akashi T; Johnson J D; Hirokawa K
Division of Clinical Pathology, Faculty of Medicine, Tokyo Medical and Dental University, Japan. nemoto-path@med.tmd.ac.jp
Pathobiology : journal of immunopathology, molecular and cellular biology (SWITZERLAND) 1997, 65 (4) p195-203, ISSN 1015-2008 Journal Code: 9007504
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Using a PCR-based cloning technique, we isolated a series of protein tyrosine kinases (PTKs) expressed in a cell line of esophageal squamous cell carcinoma. Sequence analysis revealed 10 different kinds of PTKs of the receptor type [epidermal cell growth factor receptor, insulin-like growth factor I receptor, fibroblast growth factor receptor 4, eck, erk, discoidin domain receptor (DDR)/trkE/cell adhesion kinase (Cak), HEK2, HEK8, axl and sky] and one PTK of the nonreceptor type (tyk2). Subsequently, we examined the expression of the transcripts of these 11 genes in paired samples of normal and carcinomatous esophageal tissues obtained from 12 cases of esophageal **cancer** . We found that all 11 gene transcripts were expressed in both carcinomatous and normal tissues, and 6 of them were significantly overexpressed in carcinomatous tissues relative to adjacent normal tissues. Among these, the magnitude of mRNA expression of DDR/trkE/Cak PTK was positively correlated with the proliferative activity of carcinoma cells, but not with their degree of differentiation. Immunohistochemically, DDR was expressed in both normal and **cancerous** esophageal cells. The intensity of the expression was higher in **cancer** than normal tissue. In addition, we confirmed the expression of two isoforms of DDR/trkE/Cak in normal and **cancerous** esophagus. Our study suggests that DDR/trkE/Cak plays an important role in the regulation of proliferation of esophageal **cancer** .

5/9/9 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09591911 98018855 PMID: 9357823

Epithelial cell kinase-B61: an autocrine loop modulating intestinal epithelial migration and barrier function.

Rosenberg I M; Goke M; Kanai M; Reinecker H C; Podolsky D K

Department of Medicine, Massachusetts General Hospital and Harvard Medical School, Boston 02114, USA.

American journal of physiology (UNITED STATES) Oct 1997, 273 (4 Pt 1) pG824-32, ISSN 0002-9513 Journal Code: 0370511

Contract/Grant No.: DK-41552; DK; NIDDK; DK-43351; DK; NIDDK; DK-51003; DK; NIDDK

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Epithelial cell kinase (Eck) is a member of a large family of receptor tyrosine kinases whose functions remain largely unknown. Expression and regulation of Eck and its cognate ligand B61 were analyzed in the human colonic adenocarcinoma cell line Caco-2. Immunocytochemical staining demonstrated coexpression of Eck and B61 in the same cells, suggestive of an autocrine loop. Eck levels were maximal in preconfluent cells. In contrast, B61 levels were barely detectable in preconfluent cells and increased progressively after the cells reached confluence. Caco-2 cells cultured in the presence of added B61 showed a significant reduction in the levels of dipeptidyl peptidase and sucrase-isomaltase mRNA, markers of Caco-2 cell differentiation. Cytokines interleukin-1beta (IL-1beta), basic fibroblast growth factor, IL-2, epidermal growth factor, and transforming growth factor-beta modulated steady-state levels of Eck and B61 mRNA and regulated Eck activation as assessed by tyrosine phosphorylation. Functionally, stimulation of Eck by B61 resulted in increased proliferation, enhanced barrier function, and enhanced restitution of injured epithelial monolayers. These results suggest that the Eck-B61 interaction, a target of regulatory peptides, plays a role in intestinal epithelial cell development, migration, and barrier function, contributing to homeostasis and preservation of continuity of the epithelial barrier.

5/9/10 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09325868 97237061 PMID: 9119409

ECK, a human EPH-related gene, maps to 1p36.1, a common region of alteration in human cancers .

Sulman E P; Tang X X; Allen C; Biegel J A; Pleasure D E; Brodeur G M;

Ikegaki N
Division of Oncology, Children's Hospital of Philadelphia, Pennsylvania 19104, USA.
Genomics (UNITED STATES) Mar 1 1997, 40 (2) p371-4, ISSN 0888-7543 Journal Code: 8800135
Contract/Grant No.: CA 39771; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Mouse eck, a member of the EPH gene family, has been mapped to mouse chromosome 4. The syntenic relationship between this chromosome and human chromosome 1 suggests that the human ECK gene maps to the distal short arm of human chromosome 1 (1p). Since this region is frequently deleted or altered in certain **tumors** of neuroectodermal origin, it is important to define the specific chromosomal localization of the human ECK gene. PCR screening of a rodent-human somatic cell hybrid panel by ECK-specific primers showed that ECK is indeed localized to human chromosome 1. Additional PCR screening of a regional screening panel for chromosome 1p indicated that ECK is localized to 1p36, distal to FUCA1. Furthermore, fluorescence in situ hybridization analysis with an ECK-specific P1 clone showed that ECK maps proximal to genetic marker D1S228. Taken together, the data suggest that ECK maps to 1p36.1, a region that is frequently deleted in neuroblastoma, melanoma, and other neuroectodermal **tumors** .

5/9/11 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08541055 95300106 PMID: 7780963

Protein B61 as a new growth factor: expression of B61 and up-regulation of its receptor epithelial cell kinase during melanoma progression.

Easty D J; Guthrie B A; Maung K; Farr C J; Lindberg R A; Toso R J; Herlyn M; Bennett D C
St. George's Hospital Medical School, London, United Kingdom.
Cancer research (UNITED STATES) Jun 15 1995, 55 (12) p2528-32, ISSN 0008-5472 Journal Code: 2984705R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Epithelial cell kinase (ECK) is a receptor protein tyrosine kinase, the role of which in melanoma biology is unclear. Here we studied the role of ECK during melanoma progression. ECK mRNA was overexpressed in virtually all melanoma lines tested, and levels were significantly higher in cell lines from distant metastases than primary melanomas; melanocytes were negative. Gene amplification was not detected in melanomas. Levels of ECK protein corresponded well with mRNA levels. B61 or LERK-1, recently identified as an ECK ligand, stimulated the growth of ECK-expressing melanoma cell lines, its first identified biological activity. Melanoma chemotaxis and chemoinvasion were not affected by B61. Growth of normal melanocytes was not affected. mRNA for B61 was detected in both melanoma cell lines and normal melanocytes. B61 was also identified by Western blotting and ECK binding activity with the use of a BIAcore binding assay in melanoma cell-conditioned media. These results suggest that B61 is an autocrine growth factor for melanomas but not normal melanocytes.

Tags: Comparative Study; Human; Male; Support, Non-U.S. Gov't
Descriptors: *Growth Substances--biosynthesis--BI; *Melanocytes --metabolism--ME; *Melanoma--metabolism--ME; *Membrane Proteins --biosynthesis--BI; *Proteins--biosynthesis--BI; *Receptor Protein-Tyrosine Kinases--biosynthesis--BI; *Skin Neoplasms--metabolism--ME; Blotting, Northern; Blotting, Western; Cell Division; Cell Line; Epithelial Cells; Epithelium--metabolism--ME; Infant, Newborn; Lymphatic Metastasis; Melanocytes--cytology--CY; Melanoma--pathology--PA; Neoplasm Metastasis; RNA, Messenger--analysis--AN; RNA, Messenger--biosynthesis--BI; Skin --cytology--CY; Skin Neoplasms--pathology--PA; **Tumor** Cells, Cultured; Up-Regulation
CAS Registry No.: 0 (Growth Substances); 0 (Membrane Proteins); 0 (Proteins); 0 (RNA, Messenger); 0 (endothelium secreted protein B61)

Enzyme No.: EC 2.7.1.- (**EphA2** protein); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases)

5/9/12 (Item 7 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

08489391 95242102 PMID: 7536959

Role of B61, the ligand for the Eck receptor tyrosine kinase, in TNF-alpha-induced angiogenesis.

Pandey A; Shao H; Marks R M; Polverini P J; Dixit V M

Department of Pathology, University of Michigan Medical School, Ann Arbor 48109, USA.

Science (UNITED STATES) Apr 28 1995, 268 (5210) p567-9, ISSN 0036-8075 Journal Code: 0404511

Contract/Grant No.: DK 39255; DK; NIDDK; HL 39926; HL; NHLBI; PO 1AI331890004; AI; NIAID; +

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

B61, a cytokine-inducible endothelial gene product, is the ligand for the Eck receptor protein tyrosine kinase (RPTK). Expression of a B61-immunoglobulin chimera showed that B61 could act as an angiogenic factor in vivo and a chemoattractant for endothelial cells in vitro. The Eck RPTK was activated by **tumor** necrosis factor-alpha (TNF-alpha) through induction of B61, and an antibody to B61 attenuated angiogenesis induced by TNF-alpha but not by basic fibroblast growth factor. This finding suggests the existence of an autocrine or paracrine loop involving activation of the Eck RPTK by its inducible ligand B61 after an inflammatory stimulus, the net effect of which would be to promote angiogenesis, a hallmark of chronic inflammation.

5/9/13 (Item 8 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

08448982 95197574 PMID: 7890684

Characterization of B61, the ligand for the Eck receptor protein-tyrosine kinase.

Shao H; Pandey A; O'Shea K S; Seldin M; Dixit V M

Department of Pathology, University of Michigan Medical School, Ann Arbor 48109.

Journal of biological chemistry (UNITED STATES) Mar 10 1995, 270 (10) p5636-41, ISSN 0021-9258 Journal Code: 2985121R

Contract/Grant No.: HL45351; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

B61 was originally described as a novel secreted **tumor** necrosis factor-alpha-inducible gene product in endothelial cells (Holzman, L. B., Marks, R. M., and Dixit, V. M. (1990) Mol. Cell. Biol. 10, 5830-5838). It was recently discovered that soluble recombinant B61 could serve as a ligand for the Eck receptor protein-tyrosine kinase, a member of the Eph/Eck subfamily of receptor protein-tyrosine kinases (Bartley, T.D., Hunt, R. W., Welcher, A. A., Boyle, W. J., Parker, V. P., Lindberg, R. A., Lu, H. S., Colombero, A. M., Elliott, R. L., Guthrie, R. A., Holst, P. L., Skrine, J. D., Toso, R. J., Zhang, M., Fernandez, E., Trail, G., Yarnum, B., Yarden, Y., Hunter, T., and Fox, G. M. (1994) Nature 368, 558-560). We now show that B61 can also exist as a cell surface glycosylphosphatidyl-inositol-linked protein that is capable of activating the Eck receptor protein-tyrosine kinase, the first such report of a receptor protein-tyrosine kinase ligand that is glycosylphosphatidylinositol-linked. In addition, the expression patterns of B61 and Eck during mouse ontogeny were determined by in situ hybridization. Both were found to be highly expressed in the developing lung and gut, while Eck was preferentially expressed in the thymus. Finally, the gene for B61 was localized to a specific position on mouse chromosome 3 by interspecific back-cross analysis.